Blood coagulation and fibrinolytic response after endovascular stent grafting of thoracic aorta

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Objective: **Thrombosis is common in aneurysms immediately after stent-grafting, because of exclusion from systemic blood flow. We studied changes in blood coagulation and the fibrinolytic system in patients with thoracic aortic aneurysm or dissection after stent-grafting to examine risk for consumption coagulopathy.**

Methods: **Thirty-one thoracic aortic aneurysms were treated with stent-grafting (aneurysm group), and 29 aortic dissections were treated with entry closure with stent-grafting (dissection group). The stent-graft was constructed from a self-expanding Z stent and thin-walled woven polyester fabric. Platelet count, fibrinogen, antithrombin III (AT III), and thrombin-AT III complex were assayed as markers of coagulation. Plasminogen, α_2-plasmin inhibitor, α_2-plasmin inhibitor-plasmin complex, fibrin degradation products fragment E (FDP-E), and fibrin degradation products D-dimer were monitored as markers of fibrinolysis. Blood samples were collected before surgery and on postoperative days 1, 3, 7, and 14.**

Results: **In both groups platelet count significantly decreased on postoperative days 1 and 3, and increased on postoperative day 14. AT III significantly decreased on postoperative day 1, but recovered after postoperative day 7. FDP-E significantly increased on postoperative day 1 in both groups. There was significant correlation of aneurysm diameter with α_2-plasmin inhibitor-plasmin complex, fibrin degradation products, and D-dimer in the dissection group on postoperative day 1.**

Conclusions: **Activation of coagulation and fibrinolysis was observed after stent-grafting to treat thoracic aortic aneurysm and aortic dissection. However, no patients exhibited consumption coagulopathy postoperatively. Therefore we believe there is little risk for consumption coagulopathy after stent-grafting. (J Vasc Surg 2003;37:1213-8.)**

The advantages of endovascular grafting for treatment of thoracic aortic aneurysm and aortic dissection, compared with conventional open surgery, were previously reported by the Stanford group.[1-3] However, side effects from the procedure were not sufficiently examined. Mialhe et al[4] reported that fever occurred in 57% of patients who underwent endovascular repair of abdominal aortic aneurysm, and blood coagulation disorders developed postoperatively in 10% of patients. Norgen et al[5] observed inflammatory response and decreased platelet count after endovascular grafting of aortic aneurysm. Thrombosis is commonly observed in aneurysms immediately after stent-grafting, because of exclusion from systemic blood flow.

On the other hand, enhancement of coagulation and depression of fibrinolysis occurring within 3 days after open surgery to treat abdominal aortic aneurysm (AAA) with an artificial graft has been reported.[6] Similarly, inhibition of systemic fibrinolysis and marked thrombin generation have occurred after repair of ruptured AAA, and similar changes have been noted in nonruptured AAA.[7] Brithers et al[8] found systemic fibrinolysis, detected at thrombelastography, in 25% of patients who had undergone AAA repair.[8] We expected more marked activation of coagulative and fibrinolytic systems after endovascular grafting than after open surgery, because blood products such as platelets and fibrinogen are consumed by generation of thrombus in an aneurysmal sac or false lumen.

To date there have been two reports of consumption coagulopathy after stent-grafting: Ohara et al[9] reported a case in which a patient with AAA died of adult respiratory distress syndrome and disseminated intravascular coagulation (DIC) after stent-grafting, and Cross et al[10] reported fatal consumptive coagulopathy after stent-grafting in a patient with AAA. If bleeding is prolonged, consumption coagulopathy can cause serious complications such as DIC and multiple organ failure. Differences in hemodynamics after stent-grafting have been observed between thoracic aortic aneurysm and aortic dissection. A thrombus that forms in the aneurysmal sac of a thoracic aortic aneurysm after stent-grafting is separated from systemic blood flow, whereas a thrombus in aortic dissection remains in contact with the systemic blood flow through the reentry. This suggests that activation of coagulation and fibrinolysis is more likely after stent-grafting to treat aortic dissection than after stent-grafting to treat aortic aneurysm.

We studied changes in blood coagulation and the fibrinolytic system in patients with thoracic aortic aneurysm and aortic dissection treated with stent-grafting.

METHODS

Patients. Thirty-one patients (25 men, 6 women; mean age, 69.9 years) with thoracic aortic aneurysm underwent stent-grafting (aneurysm group), and 29 patients (27 men, 2 women; mean age, 54.4 years) with aortic dissection

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Competition of interest: none.

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Table I. Patient and surgical data

	Aneurysm group (n = 31)	*Dissection group (n = 29)*	P
Age (y)	69.9 ± 7.6	54.4 ± 13.7	<.0001
Sex (% men)	80.6	93.1	NS
Diameter of aneurysm (mm)	58 ± 16	54 ± 15	NS
Operation time (min)	250 ± 74	195 ± 61	<.005
Bleeding volume (mL)	457 ± 373	168 ± 183	<.05
Blood transfusion (mL)	108 ± 197	66 ± 156	NS

Values are given as mean ± SD or percentage of patients.
NS, Not significant.

(double-barrel type) underwent entry closure with stent-grafting (dissection group). All aortic dissections were chronic (Stanford type B), without preoperative consumption coagulopathy. The results of elective stent-grafting alone are reported here; emergency cases were not included in the study. No patient in either group had a history of liver disease or was taking oral anticoagulation therapy before the operation. Anticoagulant, eg, nalmostat mesilate or gabexate mesilate (proteinase inhibitors often used to treat DIC), was not administered during the postoperative course. In all cases, before stent-grafting informed consent was obtained according to guidelines of the Ethics Committee of Tokyo Medical University.

Stent-graft. The stent-graft was constructed from a self-expanding Gianturco Z stent (Cook, Bloomington, Ind) and thin-walled woven polyester fabric (Ube Industries, Ube, Japan). The femoral artery was exposed with the patient under general anesthesia. Activated coagulation time was maintained at more than 200 seconds with intravenous administration of 50 U/kg of heparin before sheath insertion. A guide wire, 400 cm long and 0.032 inches in diameter, was introduced through the right brachial artery and down to the abdominal aorta. The distal end of the guide wire was caught with a snare catheter (Microvena, White Bear Lake, Minn) and picked up at the femoral artery. An 18F or 20F sheath (Cook) was introduced transfemorally over the guide wire with the tug-of-wire technique.[11] Excluded aneurysm was confirmed with digital subtraction angiography immediately after the operation and with computed tomography (CT) on postoperative day 10.

Blood samples. Platelet count (normal range, 140-340 $\times 10^9$/L), fibrinogen (normal range, 2-4 g/L), antithrombin III (AT III; normal, >70%), and thrombin-AT III complex (normal range, 0-2.0 ng/mL) were assayed as markers of coagulation. Plasminogen (normal range, 80%-120%), α_2-plasmin inhibitor (normal range, 80%-120%), α_2-plasmin inhibitor-plasmin complex (normal range, 0-0.8 mg/mL), FDP-E (normal, <100 ng/mL), and fibrin degradation products D-dimer (normal, <0.8 mg/mL) were assayed as markers of fibrinolysis. Samples were collected before the operation and on days 1, 3, 7, and 14 postoperatively.

Statistics. Statistical analysis was performed with StatView software (SAS Institute, Cary, NC). Differences between preoperative and postoperative values (postoperative days 1, 3, 7, and 14) were assessed with Wilcoxon signed-rank test and the Holm method. The Mann-Whitney *U* test was used to compare groups of patients. Because the data were not normally distributed, the Spearman rank test was used to assess the correlation between aneurysm diameter and various values on postoperative day 1. All values are expressed as means ± SD. $P < .05$ was considered statistically significant.

RESULTS

Surgical data (Table I). Mean aneurysm diameter was 58 ± 16 mm in the aneurysm group and 54 ± 15 mm in the dissection group. No significant difference was observed between the two groups. Stent-grafting was successfully performed in all patients. In the aneurysm group, aneurysms were completely excluded in all patients. In the dissection group, partial thrombus in the false lumen was observed on the postoperative CT scan. Postoperative change in aneurysm diameter was not noted in either group, and no graft infection, thromboembolism, or paraplegia developed in any patient. Operation time was significantly longer in the aneurysm group (250 ± 74 minutes) than in the dissection group (195 ± 61 minutes) ($P < .005$). Blood loss during stent-graft repair was significantly higher in the aneurysm group (457 ± 373 mL) than in the dissection group (168 ± 183 mL) ($P < .05$).

Changes in coagulation (Table II). Platelet count decreased significantly in both groups on postoperative days 1 and 3 ($P < .01$), but returned to the preoperative value on postoperative day 7. Platelet count in both groups on postoperative day 14 was significantly higher than the preoperative value ($P < .01$). In both groups, fibrinogen increased postoperatively, with significant increase after postoperative day 3 ($P < .01$). In contrast, AT III level decreased significantly in both groups on postoperative day 1 ($P < .01$) and recovered after postoperative day 7. Thrombin-AT III complex in both groups increased significantly on postoperative day 1 ($P < .01$) and recovered after postoperative day 7.

Changes in fibrinolytic system (Table III). Plasminogen decreased significantly in both groups on postoperative day 1 ($P < .01$), but increased after postoperative day 7, with a significant increase on postoperative day 14 ($P < .01$). In both groups, α_2-plasmin inhibitor decreased

Table II. Coagulation data

	Group	*Preoperative value*	*Postoperative day* 1	3	7	14
Platelets ($\times 10^9$/L)	Aneurysm	22.4 ± 9.2	15.6 ± 8.5†	15.3 ± 7.5†	22.7 ± 6.6	32.5 ± 9.4†
	Dissection	21.3 ± 5.0	14.9 ± 3.8†	15.4 ± 5.8†	23.4 ± 6.8	33.5 ± 9.6†
Fibrinogen (g/L)	Aneurysm	342 ± 122	332 ± 93	562 ± 183†	560 ± 172†	472 ± 108†
	Dissection	339 ± 123	365 ± 146	528 ± 165†	585 ± 197†	574 ± 177†
AT-III (%)	Aneurysm	98 ± 17	80 ± 11†	84 ± 15†	97 ± 14	99 ± 12
	Dissection	100 ± 13	91 ± 10*	95 ± 16	100 ± 12	103 ± 14
TAT (ng/ml)	Aneurysm	7.5 ± 7.4	21.1 ± 17.6†	19.9 ± 24.1†	12.4 ± 14.0	11.4 ± 15.1
	Dissection	7.3 ± 5.3	36.2 ± 18.6†	18.6 ± 16.4†	9.2 ± 4.5	9.1 ± 3.9

Values are given as mean ± SD.
AT III, Antithrombin III; *TAT,* thrombin-antithrombin III complex.
*$P < .05$ vs preoperative value.
†$P < .01$ vs preoperative value.

Table III. Fibrinolytic data

	Group	*Preoperative value*	*Postoperative day* 1	3	7	14
PLG (%)	Aneurysm	98 ± 19	81 ± 13†	87 ± 17†	110 ± 21†	111 ± 17†
	Dissection	100 ± 25	85 ± 19*	85 ± 20†	104 ± 21*	115 ± 25†
α_2-PI (%)	Aneurysm	93 ± 12	81 ± 15†	98 ± 18	104 ± 16*	95 ± 14
	Dissection	98 ± 13	82 ± 20†	98 ± 20	106 ± 17	98 ± 27
PIC (μg/mL)	Aneurysm	1.1 ± 0.5	2.0 ± 1.6†	2.1 ± 1.7†	2.3 ± 1.4†	2.4 ± 2.7†
	Dissection	2.0 ± 2.0	6.1 ± 5.5†	3.7 ± 4.5†	3.3 ± 3.8†	3.1 ± 1.9
FDP-E (ng/mL)	Aneurysm	182 ± 136	596 ± 391†	426 ± 361†	415 ± 287†	416 ± 313†
	Dissection	397 ± 69	1399 ± 1505†	694 ± 616*	572 ± 445*	499 ± 318
DD (μg/mL)	Aneurysm	5.6 ± 13.0	10.5 ± 8.1†	8.9 ± 10.1	7.3 ± 4.8	7.2 ± 5.3
	Dissection	6.8 ± 10.8	16.8 ± 14.1†	10.9 ± 8.1	9.7 ± 5.8	8.7 ± 5.2

Values are given as mean ± SD.
PLG, Plasminogen; *PI,* plasmin inhibitor; *PIC,* α_2-plasmin inhibitor–plasmin complex; *FDP-E,* fibrin degradation products fragment E; *DD,* D-dimer.
*$P < .05$ vs preoperative value.
†$P < .01$ vs preoperative value.

significantly on postoperative day 1 ($P < .01$) and recovered to the preoperative value by postoperative day 3. α_2-Plasmin inhibitor-plasmin complex increased significantly on postoperative day 1 in both groups ($P < .01$) and on postoperative day 14 in the aneurysm group ($P < .001$). FDP-E increased significantly on postoperative day 1 in both groups ($P < .01$). In the dissection group, no further significant increase in FDP-E was observed after postoperative day 3; however, in the aneurysm group FDP-E continued to increase after postoperative day 3 ($P < .01$). D-Dimer increased significantly in both groups on postoperative day 1 ($P < .01$), and no significant increase was observed after postoperative day 3.

Differences in coagulation and fibrinolytic system between groups (Figs 1 and 2). There were no significant differences in platelet count, fibrinogen, AT-III, plasminogen, α_2-plasmin inhibitor, FDP-E, or D-dimer between groups. A significant difference in thrombin-AT III complex level was observed on postoperative day 1 ($P < .005$).

On postoperative day 1 the aneurysm group had significantly lower levels of α_2-plasmin inhibitor-plasmin complex compared with the dissection group ($P < .005$).

Correlation of aneurysm diameter with coagulation and fibrinolytic system (Table IV). Aneurysm diameter correlated significantly with α_2-plasmin inhibitor-plasmin complex, FDP, and D-dimer in the dissection group on postoperative day 1. No other significant correlations were observed.

DISCUSSION

Recent advances in endovascular therapy have been remarkable. In particular, developments in stent-graft technology for treatment of aortic aneurysm or dissection have made it an intriguing option. Conventional open surgery to treat thoracic aortic aneurysm or dissection is an invasive cardiopulmonary bypass procedure. In contrast, stent-grafting is a minimally invasive procedure with short operation time and little blood loss. However, few reports have addressed the complications associated with stent-grafting, including fever and postoperative decrease in platelet count,[4,5] as well as fatal sequelae such as coagulative disorders.[9,10]

Hollier et al[12] reported nonresective therapy for AAA in patients at high-risk. In their study, abdominal aneurysm was excluded by ligating the proximal and distal sides of the

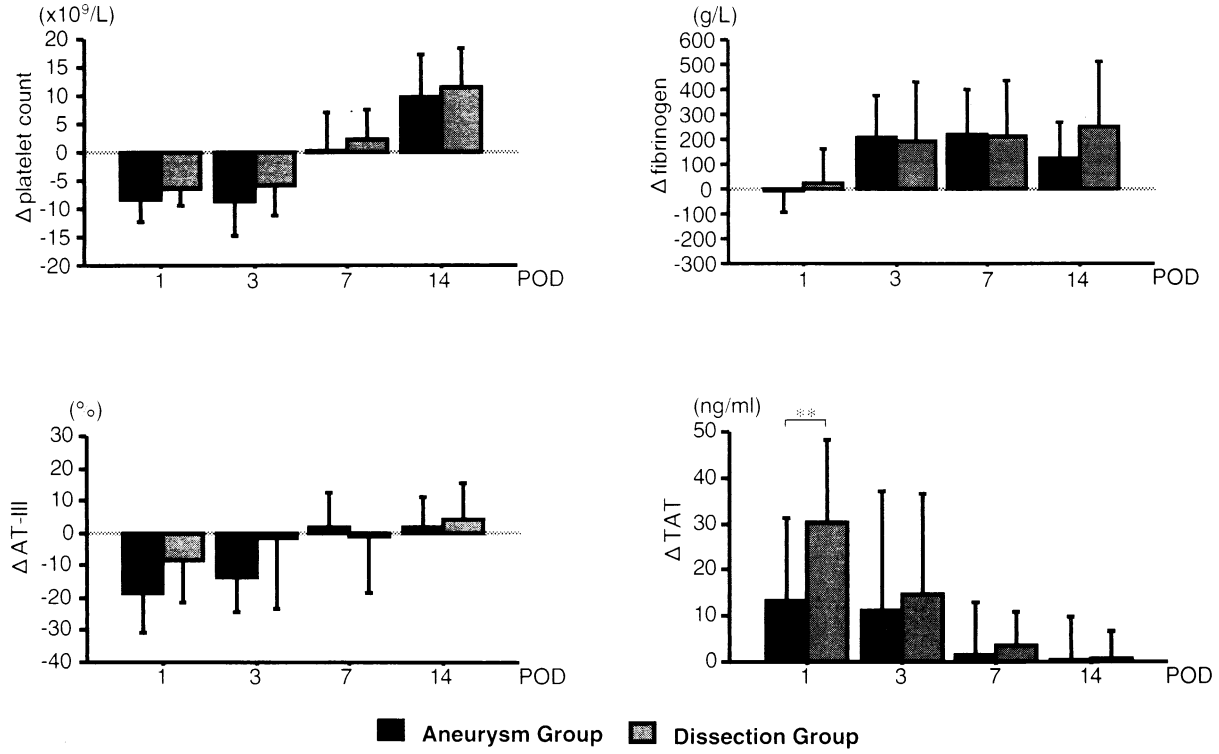

Fig 1. Differences between preoperative and postoperative coagulation indices. Δ, Difference between preoperative value and each postoperative value. $*P < .05$, $**P < .01$ for comparison of changes between aneurysm group and dissection group. *POD,* Postoperative day; *AT-III,* antithrombin III; *TAT,* thrombin-antithrombin III complex.

aneurysm with an axillobifemoral bypass. Schwartz et al[13] reported the possibility of DIC as a complication of this type of nonresective therapy for aortic aneurysm. Therefore we considered the possibility of consumption coagulopathy due to thrombus formation in the aneurysm sac after stent-grafting. We checked for a decrease in AT III and an increase in thrombin-AT III complex, because these are involved in the last steps of the transformation of fibrinogen to fibrin. Plasmin is an important element of the fibrinolytic system, but it is difficult to measure plasmin because of its short period of activity. Therefore we measured α_2-plasmin inhibitor, an inhibitor of coagulation, and α_2-plasmin inhibitor-plasmin complex, a complex of plasmin and α_2-plasmin inhibitor. We found acceleration of coagulation and the fibrinolytic system on postoperative day 1; however, all factors began to return to preoperative levels on postoperative day 3. Also, none of our patients exhibited consumption coagulopathy preoperatively or postoperatively.

Ohara et al[9] reported a patient with AAA and severe liver cirrhosis who died of ARDS and DIC after stent-grafting. In that case, DIC resulted from acute consumption of coagulation factors, and ARDS resulted from high levels of cytokines such as interleukin-6 and tumor necrosis factor-α; these cytokines were not inactivated, because of severe liver dysfunction. Preoperative platelet count in that case was 55×10^9/L, suggesting that the likelihood of consumption coagulopathy is higher in patients with a preoperative coagulation disorder. Cross et al[10] reported development of consumptive coagulopathy after stent-grafting. They found that the endothelium was stimulated by endovascular devices and speculated that the microembolism, which occurred as petechial haemorrhage involving the skin and the gastric and vaginal mucosae, caused consumption coagulopathy by activating coagulative cascades. In that case no preoperative coagulation disorder was found, but the stent- grafting procedure was technically difficult and time-consuming because of the small, tortuous iliofemoral vessels. Thus it appears that the likelihood of consumptive coagulopathy is higher in cases in which there is a preoperative coagulative disorder or stent-grafting is technically difficult.

The changes we observed in platelet count were of great interest. Platelet count decreased on postoperative day 1 but recovered by postoperative day 7. On postoperative day 14 the count was higher than the preoperative value. Such biphasic changes have been reported after replacement of an infrarenal AAA with a vascular prosthesis. Utoh et al[14] reported that the time to peak platelet count varied with artificial blood vessel type. The stent-graft in our study was constructed from a Z stent and thin-walled

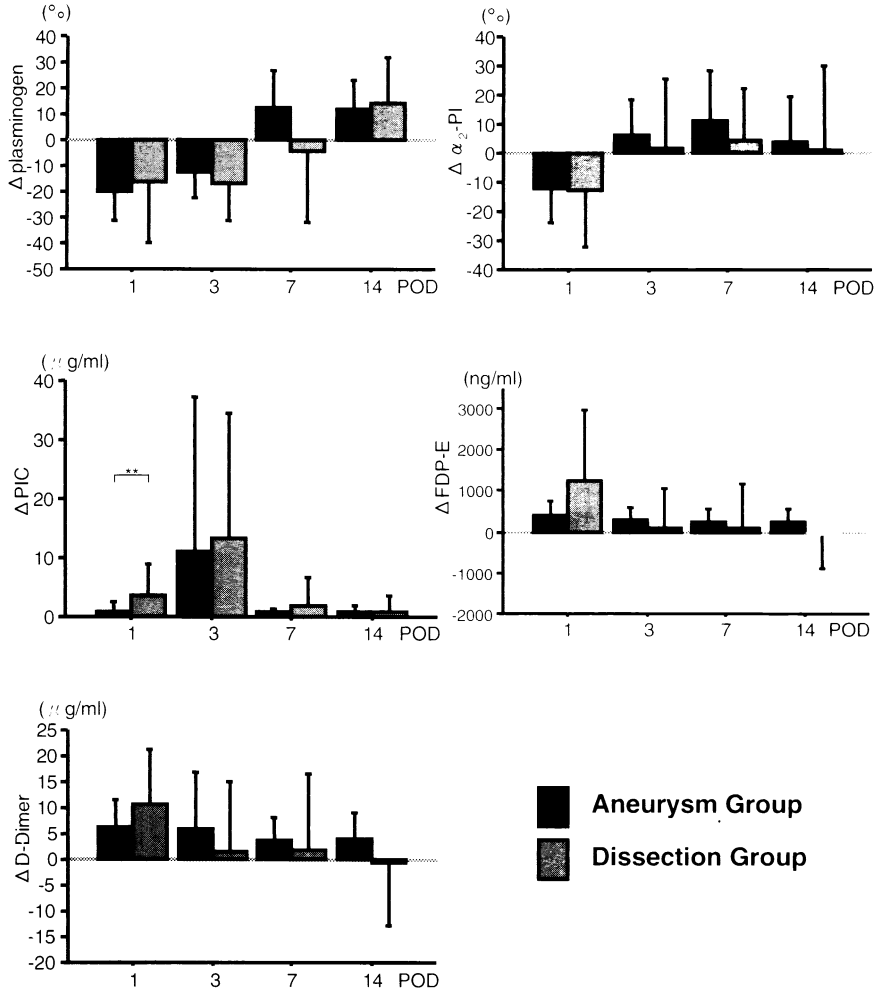

Fig 2. Differences between preoperative and postoperative values in fibrinolytic system. Δ, Difference between preoperative value and each postoperative value. *P < .05, **P < .01 for comparison of changes between aneurysm group and dissection group. *PI,* α_2-Plasmin inhibitor; *PIC,* α_2-plasmin inhibitor-plasmin complex; *FDP-E,* fibrin degradation products E fragment.

woven polyester fabric. Further study is required to clarify the effects of various vascular prostheses on platelet count.

There were significantly larger increases in thrombin-AT III complex and α_2-plasmin inhibitor-plasmin complex in the dissection group than in the aneurysm group on postoperative day 1. We considered the possible causes of these differences. There was a clear difference in purpose of performing stent-grafting between the two groups, ie, to exclude aneurysm and prevent rupture of aneurysm in the aneurysm group and to close the intimal tear and decompress the false lumen in the dissection group. Therefore retrograde blood flow to the false lumen occurred through reentry in the dissection group, whereas blood flow in the aneurysm group was antegrade. The difference in hemodynamics on the various postoperative days may have produced the differences in results between the aneurysm group and the dissection group, but other factors may also be involved.

Table IV. Correlation of aneurysm diameter with coagulation and fibrinolytic data

	r	
	Aneurysm group	*Dissection group*
Platelets	0.22	0.06
Fibrinogen	−0.04	−0.12
AT-III	0.05	−0.38
TAT	−0.12	0.13
Plasminogen	−0.16	−0.23
α_2-PI	−0.05	−0.35
PIC	0.18	0.62*
FDP-E	0.17	0.69*
DD	0.29	0.57*

AT-III, Antithrombin III; *TAT,* thrombin–antithrombin III complex; *PI,* α_2-plasmin inhibitor; *PIC,* α_2-plasmin inhibitor–plasmin complex; *FDP-E,* fibrin degradation products fragment E; *DD,* D-dimer.
*$P < .01$; significant correlation.

In the dissection group we found significant correlation of aneurysm diameter with α_2-plasmin inhibitor-plasmin complex, FDP-E, and D-dimer on postoperative day 1. In patients with a large false lumen, α_2-plasmin inhibitor-plasmin complex, FDP-E, and D-dimer increased on postoperative day 1, and a hyperfibrinolytic state was observed. A large false lumen results from torrential flow through a large primary tear into the false lumen. In cases with a large false lumen after stent-grafting, there is considerable retrograde blood flow to the false lumen and intense turbulent blood flow. It is important to measure coagulation and fibrinolytic factors in patients with aortic dissection with a large false lumen, because these patients are in a hyperfibrinolytic state. We could not find a significant correlation between aneurysm diameter and coagulation factors. Therefore we are not certain whether the hyperfibrinolytic state causes DIC. We did not find a correlation in the aneurysm group between aneurysm diameter and coagulation or fibrinolytic factors on postoperative day 1. This may have resulted from cutting off the thrombus in the aneurysm from the systemic blood flow and preventing thrombus formation in the venous blood. Therefore the possibility of consumption coagulopathy is not increased, even if stent-grafting is performed to treat large true aneurysms.

Comparison with conventional surgery is required to further clarify the changes in the coagulation and fibrinolytic factors after stent-grafting to treat thoracic aortic aneurysm. However, in conventional open surgery, extracorporeal circulation and high doses of heparin are required for continuous blood flow to the main organs. In addition, blood loss increases, sometimes resulting in homogeneous blood transfusion. Thus comparison with stent-grafting may be difficult, because other factors influence coagulation and fibrinolysis.

Because the present study is a negative study, we must consider the risk for type II error. We performed stent-grafting in more than 180 patients with thoracic aneurysm and more than 80 patients with aortic dissection, and consumptive coagulopathy did not develop in any patients. Therefore we were unable to determine the rate of occurrence of this side effect. This qualifies as a limitation of the study. Accordingly, we will be on the lookout for future case reports of consumptive coagulopathy as a side effect of stent-grafting.

In conclusion, activation of coagulation and fibrinolysis was observed after stent-grafting to treat thoracic aortic aneurysm and aortic dissection. However, none of our patients had consumption coagulopathy postoperatively. Therefore we believe that there is little risk for consumption coagulopathy after stent-grafting. We recommend measurement of the markers of coagulation and the fibrinolytic system in patients with aortic dissection with a large false lumen, because fibrinolysis is associated with false lumen diameter.

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